

SUPPORTING DOCUMENT ON MAEDI-VISNA

1. Introduction

Ovine lentiviruses, maedi-visna virus (MVV) and South African ovine maedi-visna virus (SA-MVV), can infect sheep causing maedi-visna disease (MV) (Banks *et al*, 1983). The ovine lentiviruses are closely related to, but genetically and serologically distinct from, caprine arthritis encephalitis virus (CAEV) (Pasick 1998; Valas *et al*, 2000).

2. Current world situation

It is difficult to assess the prevalence of MV globally because in some countries the disease is not reported. This may be due to the nature of the disease, which is usually latent and expressed mainly in older animals. Some under-reporting of MV may be due to alternative names for the disease; ovine progressive pneumonia, Montana sheep disease, zwoegersiekte, la bouhite, lungers, Marsh's progressive pneumonia and Graaff-Reinet disease.

3. Clinical signs

MV is a slowly progressive, insidious disease of sheep usually manifested either in the respiratory, or central nervous system (CNS). Experimental infection can result in acute disease in young animals with very high mortality (Andresson *et al*, 1993).

Differential diagnosis

The disease must be differentiated from other causes of chronic respiratory and nervous system disease, including pulmonary adenomatosis, parasitic pneumonia, chronic bacterial pneumonia, scrapie, listeriosis, pregnancy toxemia, plant poisoning and parasitic CNS invasion, eg *Coenuris cerebralis*.

4. Pathology

Lymphoproliferation caused by MVV may affect the lungs, mediastinal lymph nodes, brain, joints and mammary glands (Verwoerd *et al*, 1994). Demyelination in the presence of leucoencephalomyelitis is common in the central nervous system. The lymphocytic component of the inflammatory infiltrates is thought to be responsible for the observed neurological damage (Sanders *et al*, 2001). Not all strains of MVV lead to progressive encephalopathy (Campbell and Robinson, 1998).

5. Epidemiology

Spread

Sihvonen *et al* (1999) warns that if introduced into a free country, MV can spread widely before clinical cases are detected. This has happened in Iceland, Sweden and Finland (Fridriksdottir *et al*, 2000; Hugoson, 1978; Sihvonen *et al*, 2000). In Sweden the disease was first recognised in 1974 and by 1975, a limited survey revealed 23

flocks were positive (Hugoson, 1978). The introduction of MV into Finland was traced to the importation of infected seronegative sheep in 1981 (Sihvonen *et al*, 1999).

Course of infection

MVV infections are characterised by a long and variable incubation period and life-long viral persistence (Cutlip *et al*, 1988) and clinical signs are rarely seen in sheep less than 3 years old (Constable *et al*, 1996). The antibody response confers no resistance to disease and the clinical course of disease is progressive (Carey and Dalziel, 1993; Verwoerd *et al*, 1994).

Viraemia develops shortly after infection and plays a major role in distribution of monocyte associated virus throughout the body (Georgsson, 1990).

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Prevalence

Studies in Canada, the USA and some EU countries have shown the average flock seroprevalence of MV can range from 19% to 97% (Constable *et al*, 1996; Lujan *et al*, 1993; Houwers *et al*, 1987).

Lateral transmission

The target cells for MVV replication are mononuclear cells and transmission of virus occurs via these cells (Joag *et al*, 1996). Transmission predominantly occurs from ewe to lamb via ingestion of colostrum (Sihvonen, 1980). Lateral transmission can also occur during close contact, mainly via respiratory secretions. This form of spread is enhanced if an animal is coinfectd with other pulmonary infections particularly pulmonary adenomatosis.

Vertical transmission – via embryos

The evidence for transplacental transmission of MVV is equivocal. Preventing colostral transfer and early contact with infected dams has been regarded as an effective means of obtaining MV free progeny (De Boer *et al*, 1979; Cutlip *et al*, 1988; Sihvonen, 1980). Long-term absence of MVV infection was demonstrated in a group of approximately 40 lambs separated from infected ewes immediately after birth and reared in isolation (De Boer *et al*, 1979). Similar results were reported by Light *et al* (1979) and Houwers *et al* (1987). Other studies suggest that the potential for transplacental infection cannot be entirely dismissed. Cutlip *et al* (1981) reported prenatal transmission based on the detection of MVV from 1 foetus and 2 newborn lambs out of 70 progeny. Cross *et al* (1975) reported infection in a small proportion of hysterectomy derived lambs from infected dams. More recently, Brodie *et al* (1994) detected MVV DNA in the peripheral blood mononuclear cells (PBMC) of 11% of lambs removed from their infected dams immediately after birth.

Viraemia which develops shortly after infection might expose embryos to virus. Using PCR techniques, Woodall *et al* (1993) failed to detect MVV in either uterine washes or washed embryos collected from 10 infected ewes. Further studies, involving increased numbers of animals at different stages of infection, are required to conclude that exposure of embryos to MVV during infection does not occur.

Vertical transmission – via semen

Transmission of infection via semen has not been demonstrated (Dawson 1987). However, ovine lentivirus was detected in the semen of rams concurrently infected with *Brucella ovis* (de la Concha-Bermejillo *et al*, 1996). These authors suggest that inflammatory lesions of the genital tract causing leucocytospermia, as caused by *B ovis*, predispose infected rams to shed ovine lentivirus in their semen. Moreover semen may contain blood or plasma and MVV capsid antigen has been detected in plasma of infected sheep (Brodie *et al*, 1994). These studies do not provide clear

evidence that MVV is transmitted to recipient ewes or offspring via infected semen but do suggest the potential for venereal transmission.

Breed susceptibility

Differences in breed susceptibility to MVV have been reported (Houwens *et al*, 1989). Icelandic breeds appear to be more susceptible than British breeds and Texels and Border Leicester are more susceptible to disease than Columbia sheep (Cutlip *et al*, 1986; Joag *et al*, 1996). Also, Snowden *et al* (1990) determined significant differences in the seroprevalence of MV between the 6 breed types comprising a flock of 2,976 sheep. Nevertheless, complete breed-associated resistance has not been demonstrated (Houwens, 1990). Houwens *et al* (1989) suggest that apparent susceptibility may also depend on the strain of MVV.

Host range

Disease due to MVV has only been reported in sheep and very rarely in goats (Castro *et al*, 1999; Banks *et al*, 1983).

6. Adverse consequences of MVV

A significantly lower reproduction rate was observed in seropositive ewes and their lambs suffered from significantly higher death and lower growth rates, probably due to a reduced milk production, resulting in economic losses (Scheer-Czechowski *et al*, 2000). This observation contrasts with that of Dungu *et al* (2000) who reported minimal difference between the pre-weaning growth of lambs born of ewes naturally infected with South African strains of maedi visna virus (MVV) and uninfected ewes kept under similar conditions.

In general, introduction of the MVV into a free country or zone results in an adverse economic impact. In most situations the disease causes significant losses due to deaths, 'ill thrift' and the cost of control and eradication measures. In recognition of this adverse impact eradication programs have been implemented in the Netherlands (Houwers, 1990), Canada (Williams-Fulton and Simard, 1989), Iceland (Zanoni *et al*, 1994), Finland (Sihvonen *et al*, 1999), Sweden (Lindqvist, 1994), USA (Young, 1993) and Germany (Scheer-Czechowski *et al*, 2000).

7. Risk management

a) Disease freedom of animals in country, region or flock

Country/zone freedom

Reporting country status to the OIE with respect to MV is currently unreliable (Brodie *et al*, 1994; Constable *et al*, 1996; HandistatusII). Reliance on country or zone freedom as an effective risk management option therefore requires the specification of extra measures to ensure that a country or zone claiming freedom has adopted strategies to ensure this to be the case. MV should be notifiable in the whole country, and all clinical cases suggestive of MV should be thoroughly investigated. If serosurveys are not conducted, measures to prevent the introduction of the disease via animals or their genetic products should have been in place for at least 5 years. This period of time is expected to allow expression of disease if present in flocks before controls were implemented.

Flock freedom

Because serological testing is not always reliable and the disease has a long latency period, assurances of flock freedom within an infected country or zone may be difficult. Johnson *et al* (1992) observed that the absence of clinical signs over a 5 year period alone can not be regarded as evidence of flock freedom. Similarly, Williams-Fulton and Simard (1989) advise that a longer time period than 4 years is required to ensure that MV has been completely eradicated from a flock. Houwers (1990) recommends certification of MVV freedom for flocks based on recent serological examination of the whole flock with negative results. Continuous surveillance was found to be necessary during the eradication

program in the Netherlands. This was expected due to the delay or absence of seroconversion in some infected animals (Houwers *et al*, 1987).

Sihvonen *et al* (1999) advise that surveillance of MV has to be continuous, requiring extensive, repeated serological testing and restrictions on the movement of sheep between flocks.

b) Embryo washing

Limited studies indicate that MVV does not transmit from infected sheep through transfer of embryo (Dawson and Wilmot 1988; Young 1993). IETS (1998) regard this disease agent as Category 4 in sheep (that is, “Diseases or disease agents on which preliminary work has been conducted or is in progress”).

c) Testing and examination

Clinically normal infected animals may be detected by serology or virus isolation, however both techniques can be unreliable.

Appendix XV (contd)

Period from infections to antibody development

The period between exposure to virus and the detection of antibodies varies with the route of infection, form of exposure and breed of sheep. Seroconversion occurs from 4 to 6 weeks following experimental infection and antibody levels tend to stay relatively constant (Petursson 1990). The first appearance of antibodies following natural infection can range from 11 months to over 5 years (Houwens *et al*, 1987). Persistent high antibody titres are usual in infected animals but disease in the absence of positive serology has been described (Houwens *et al*, 1987). A complicating factor is that significant viral antigenic variation can occur in MVV infected animal over time (Narayan *et al*, 1977). Also, the serological response to MVV varies with age and breed of sheep (Constable *et al*, 1996).

Sihvonen *et al* (1999) documents the failure of quarantine measures to prevent introduction of the disease into Finland. Introduction of infected seronegative sheep in 1981 were thought to be responsible for introduction of the disease, detected 13 years later during serosurveillance (Sihvonen *et al*, 1999).

Age effects

Viral RNA can be detected in PBM cells taken from naturally infected lambs less than 1 year of age by *in situ* hybridisation. However, animals less than 1 year of age rarely show seropositivity when infected (Johnson *et al*, 1992) and an increased seroprevalence occurs with age (Simard and Morley, 1991). Snowden *et al* (1990) determined the average seroprevalence to be 11% at one year of age and 93% in sheep 7 years or older. Cutlip *et al* (1992) found that prevalence increased from 4% at less than 1 year to 34 % at 4 years, with variability associated with breed type.

Available serological tests

The agar gel immunodiffusion (AGID) test and the enzyme linked immunosorbent assay (ELISA) are the most commonly used serological tests (Simard and Briscoe 1990). The sensitivity of both tests is dependent on the antigen used (Knowles, 1997; Rosati *et al*, 1994; Saman *et al*, 1999).

Other detection methods

MV virus can be detected by virus isolation or nucleic acid detection methods. Even though virus cannot be recovered directly from tissue homogenates virus can be detected if explanted or by co-cultivating with a permissive cell type (Carey and Dalziel 1993).. A number of polymerase chain reaction (PCR) assays have been described which detect MV DNA in infected tissues, especially bone marrow, PBM cells and pulmonary leucocytes (Brodie *et al*, 1992; Celer *et al*,

2000; Johnson *et al*, 1992). (A PCR test has been used to detect infected sheep in the Dutch National MVV/CAEV control program (Wagter *et al*, 1998).

Summary – the reliability of serological testing for detecting infected animals

Viral infections are characterized by a window period during which the host is infected but diagnostic test (eg. antibody) results are negative. Animals determined to be infected by *in situ* hybridisation, PCR and co-cultivation were negative on serology (Johnson *et al*, 1992). Infection of other animals can occur during this period of seronegativity. To detect infections reliably, it is important to conduct antibody tests after the host animal has been given sufficient time to mount a detectable immune response. Houwers *et al* (1987) based accreditation of flocks on testing for MVV antibodies twice with an interval of 6 months. However, eradication efforts in Finland relied on five consecutive serological tests at 12 to 16 month intervals. Repeated testing of all animals over 1 year of age was determined to be necessary because of the lack of sensitivity of serological testing. It was reasoned that if infected animals were missed, transmission would occur and eventually produce seropositive animals within the flock (Sihvonen *et al*, 2000).

In summary, the time required for seroconversion following infection can be relatively prolonged and unpredictable. An infected animal may give a negative result to a single antibody test so more than one test over a period, and reliance on flock testing rather than individual animal testing would be expected to increase the likelihood of sourcing non-infected animals.

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CHAPTER 2.4.5.

MAEDI-VISNA

Article 2.4.5.1.

Country or zone free from maedi-visna

A country or zone may be considered free from maedi-visna (MV) if:

- 1) it has a record of regular and prompt disease reporting in all livestock;
- 2) it has reported no clinical, epidemiological, serological or other evidence of MV during the past 5 years;
- 3) MV is notifiable in the whole country, and all clinical *cases* suggestive of MV are subjected to field and laboratory investigations;
- 4) all imports of sheep (except for slaughter) from other countries or zones over the past 5 years originated from an MV free country, zone or flock;
- 5) all sheep semen and embryos/ova imported for the past 5 years met the requirements referred to in Article 2.4.5.5. and in Article 2.4.5.6., respectively.

Article 2.4.5.2.

Flock free from maedi-visna

A flock may be considered free from MV if:

- 1) it is present in a country or zone which has a record of regular and prompt disease reporting in all livestock;
- 2) no clinical, post-mortem, serological or other evidence of MV has been found in any animal in the herd during the past 5 years;
- 3) MV is notifiable in the whole country, and all clinical *cases* suggestive of MV are subjected to field and laboratory investigations;
- 4) all sheep over 3 years of age have tested negative for MV annually for at least 5 years;
- 5) all sheep introduced into the flock over the past 5 years originated from an MV free country, zone or flock;
- 6) all sheep semen and embryos/ova introduced into the flock for the past 5 years fulfilled the requirements referred to in Article 2.4.5.5. and in Article 2.4.5.6., respectively.

Appendix XVI (contd)

Article 2.4.5.3.

Veterinary Administrations of importing countries should require:

for sheep for breeding or rearing

the presentation of an *international veterinary certificate* attesting that the animals:

- 1) come from a country or zone free from MV, or
- 2) come from an MV free flock.

Article 2.4.5.4.

Veterinary Administrations of importing countries should require:

for sheep for slaughter

the presentation of an *international veterinary certificate* attesting that the animals:

- 1) are not being exported as part of an eradication programme;
- 2) showed no clinical sign of MV on the day of shipment.

[Note: Appropriate precautions should be taken both by the exporting country and the importing country to ensure that the sheep are transported directly from the place of shipment to the abattoir for immediate slaughter.]

Article 2.4.5.5.

Veterinary Administrations of importing countries should require:

for ovine semen

the presentation of an *international veterinary certificate* attesting that:

- 1) the donor animals were resident for a minimum period of 5 years immediately prior to the time of semen collection in an MV free country, zone or flock;
- 2) the semen was collected, processed and stored in conformity with the provisions of Appendix 3.2.2.

Article 2.4.5.6.

Veterinary Administrations of importing countries should require:

for ovine embryos/ova

the presentation of an *international veterinary certificate* attesting that:

- 1) the donor females were resident for a minimum period of 2 years immediately prior to the time of embryo collection in an MV free country, zone or flock;
 - 2) the embryos/ova have been collected, processed and stored in conformity with the provisions of Appendix 3.3.2.
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